like that in general paresis. In general paresis, however, we may find all sorts of tremors, from the finest to the coarsest. By itself, it is simply suggestive of the disease, and we must look for other symptoms.

Dr. PETERSON said he has seen a number of cases of neurasthenia in which the facial tremor was present; he has also observed it in diffuse syphilis of the central nervous system.

Dr. STARR confirmed the statement that tremor of the face is not very infrequent in severe types of neurasthenia, and that it is not to be relied upon as a diagnostic symptom of general paresis.

Dr. DANA, in closing the discussion, said that it is very rare indeed to get a facial tremor in any case of neurasthenia, no matter how severe, when there is no tension of the facial muscles; that is, when the muscles are at rest. A tremor of the lips, for instance, without any tension of the muscles, he would certainly consider as an evidence of serious trouble.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 24, 1892.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. WM. H. MORRISON read a paper entitled

REPORT OF A CASE OF LARGE INTRA-CRANIAL TUMOR (WEIGHING FIVE OUNCES) COMPRESSING THE LEFT FRONTAL LOBE.

A. C., colored, about forty years of age, came under the author's observation in April, 1886. At this time he was a coachman by occupation, but had been in the army and had also served as a sailor, and was said to have suffered shipwreck on one or two occasions. He complained of pains in the head, which were attributed to localized syphilitic meningitis, and he was placed upon iodide of potassium. He soon passed from the author's observation, and was not seen again until two days before death. In the summer of 1888 he had an epileptiform convulsion, which was the only convulsion in the history of the case. The pains in the head returned and increased gradually in severity, and were especially marked on stooping. In August, 1889, he gave up his work, but for a month before this his friends had noticed that his speech was slower than usual. Shortly after quitting work his eyesight began to fail, and soon he was unable to recognize persons or objects at a short distance from him. The pain seemed to be more in the occipital region, although on stooping there would be pain felt in the frontal region. During the last six or nine months of the history the pain was not so severe, and at no time does it seem to have been a marked symptom. A few months after quitting work he was admitted to the Pennsylvania Hospital, where he remained six weeks.

An examination of the eyes showed no light-perception. In the right eye there is extensive hemorrhagic retinitis, with superficial and deep hemorrhages scattered over the fundus, rendering it difficult to see the disc. The margin of the disc is obliterated. There is extensive œdema. Near the macula are several yellowish white patches, suggestive of hemorrhage. The condition of the left eye is about the same.

After leaving the hospital he received no medical attention. The principal trouble observed by the patient and his friends was the loss of eyesight, which had sunk to simple perception of light. During the day he sat about the house, and at night he was able, unaided, to go up three flights of stairs. In fact, a few days before his death, he dressed himself and came downstairs without assistance. There was no motor disturbance in any part of the body—no weakness of the muscles. Intelligence did not seem to be materially impaired. His memory for past events was retained. In illustration of this it may be stated that two weeks before his death he was visited by friends whom he had not seen for years, and conversed with them intelligently and rationally about events that had occurred ten or more years previously. For recents events, however, memory was defective. The drawling speech continued, and to this was added slight amnesic aphasia. Occasionally he would be unable to remember a certain common word which he wished to use, but when it was suggested he would recognize it and be able to pronounce it without difficulty. Hearing was unaffected; the sense of smell was good, but whether or not there was any difference on the two sides could not be ascertained; the bowels and bladder acted normally.

The writer was called to see him at midnight on December 24, 1891, the statement being that he was in a stupor from which he could not be roused. When seen, however, he was awake. He protruded the tongue normally. He lifted first one arm and then the other, and moved the legs at command. He had high fever, injected conjunctivæ, and other symptoms of influenza, which was prevalent at that time. The following day his condition was about the same; but next day he passed into a comatose condition, and died that evening.

The autopsy was made fourteen hours after death, the examination being confined to the head. The body was that of a stout, well-nourished man, with nothing abnormal on inspection. The scalp was fully one-half inch in thickness, there being a quarter-inch layer of fat; the bone was of normal thickness, and the dura but slightly adherent to its under surface; the dura was found strongly adherent to the pia mater along each side of the longitudinal fissure, and a portion of it was removed with the brain. A large tumor was at once evident in the left frontal region. The brain and tumor were removed as carefully as possible. It was found that the tumor had eroded the left half of the cribriform plate of the ethmoid, so that there was a direct communication between the interior of the skull and the nasal cavity.

From the inner surface of the frontal bone, one inch to the left of the median line and one inch above the orbital plate, there sprang a conical projection of bone; this had a base one-half inch in diameter, and projected three-eighths of an inch above the surface of the bone. Corresponding to this exostosis, there was a depression in the dura mater and in the new growth. Inspection of the external surface of the bone showed no depression and no evidence of injury.

Examination of the brain and tumor, after removal, showed that the growth had occupied the greater part of the left anterior fossa of the skull. The mass is irregularly pyramidal in shape, its base, corresponding to the left half of the frontal bone, being attached to the inner surface of the dura. The base of the growth measures two and one-half inches vertically by three and one-quarter inches transversely. The tumor extends backward into the frontal lobes two and three-quarter inches. After preservation in alcohol for five months, the weight of the mass is five ounces, its volume nine cubic inches, and it displaces five fluid ounces of water. The orbital plate of the frontal bone was not displaced, and there was no exophthalmus. The tumor was not adherent to the brain-tissue, but seems to be a dural tumor originating at the position of the projection of bone already mentioned, and thence extending gradually, pressing backward the frontal convolutions. Internally and externally the tumor was covered by cerebral tissue; but underneath, the brain-tissue has been displaced and absorbed in the position of the cribriform plate, and the bone itself has been destroyed. The tumor was readily lifted from its cavity, and, in fact, care was necessary to prevent it from falling out when the brain was removed.

The growth was examined microscopically by Dr. C. W. Burr, who pronounced it to be a sarcoma.

The special features of this case, as elicited by post-mortem study, are: The evidences of a slowly-developing and steadily-progressing lesion, accompanied by pain in the head, not distinctly localized, not especially severe; a single epileptiform convulsion, neuro-retinitis, loss of memory, slowness of mental processes, slowness of speech, and slight amnesic aphasia. To these may be added absence of motor disturbances, and absence of signs of involvement of the special senses other than the sight.

Referring to lesions in this region, Dr. Seguin says:[1]

"Focal lesions of the frontal lobe produce no specific symptoms, and cannot be directly diagnosticated unless they extend as far caudad as the base of the second or third frontal gyri. The forward mass of the frontal lobe including the orbital lobule appears to be inexcitable and insensitive. Even psychic symptoms do not necessarily appear after the loss of a considerable amount of cerebral substance from this region. The diagnosis of tumors, abscesses, etc., in this part of the brain must be made by taking into consideration the seat of the pain, the presence of cicatrices or other etiological indications, the general signs of cerebral irritation and compression, but, after all, usually by exclusion. In some cases unilateral anosmia is produced."

Dr. Mills, speaking of lesions in this part of the brain, says:[2]

"Lesions of the prefrontal lobe, although this is one of the so-called latent districts of the brain, have, in a large percentage of the carefully-studied cases, shown distinct manifestations. The symptoms are largely psychical, and, unfortunately, the physician is not usually well trained to study such phenomena. Mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason. Closely studied, the inhibitory influence of the brain both upon psychical and physical action is found to be diminished. Memory is not seriously affected, although a continuous train of thought cannot well be followed, and complex intellectual processes cannot be thoroughly performed."

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[1] Pepper's System of Medicine, vol. v., p. 89.

[2] Transactions of the Congress of American Physicians and Surgeons, vol. i., p. 272.

A review of the history of this case, as thus imperfectly elicited, seems to indicate that, had the patient been under careful observation, a diagnosis of the probable seat of the growth would have been made, and, possibly, with sufficient certainty to have rendered an exploratory opening justifiable. Could the tumor have been located some months or a year before death, its removal would have been comparatively easy.

Dr. Ferrier, in commenting upon a case, very similar to the one reported in this paper, in regard to tumors in this situation, says:[3] "It is not until the tumor presses backward and, directly or indirectly, disturbs the functions of the central and posteriorly situated convolutions, that motor or sensory symptoms declare themselves; and until these have occurred, or until there are signs of implication of the nerves or other structures in the anterior fossa, an accurate diagnosis of the position of the tumor cannot be made with certainty, inasmuch as loss of memory and impaired power of concentration may also be produced by diffuse lesions or lesions which cause general disturbance of the brain. Of the structures in the anterior fossa, the olfactory tract is especially apt to suffer, but, on account of our patient's mental condition, it never could be ascertained with certainty whether there was any implication of smell in the one nostril or the other. During life, however, I had expressed the opinion that the case was in all probability one of tumor of the frontal region, but its precise position, whether deeply seated or superficial, or connected with the corpus callosum, did not appear to me sufficiently clear. For this reason, though I frequently considered the advisability of operation, I did not think it well to interfere until definite localizing symptoms should declare themselves; but the patient was carried off by an intercurrent malady before this event occurred. The necropsy, however, proved that the tumor might have been reached and, in all probability, enucleated by trephining over the left frontal lobe anteriorly."

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[3] Lancet, June 4, 1892.

DISCUSSION.

Dr. JAMES HENDRIE LLOYD.—About a year ago I put on record a case of tumor in the frontal lobe not exactly in this situation. That case had been successfully diagnosticated, and the tumor had been localized. A consultation was held to consider the propriety of trephining; but the man developed tuberculosis of the lung, to which he succumbed rapidly, and the operation was not done. In that case the most prominent symptom, and one which I have never seen in any other brain-lesion, was the peculiar inhibition of thought—the increase of the time-reaction of cerebration. Although Dr. Ferrier, in the communication referred to to-night, says that it may be present as a symptom of other lesions, I have never seen it in any other lesion so characteristic and so typical. In the case which I reported, the man was intelligent and answered questions properly; but it took him a long time to do so, as though, to use a simile, the question had to percolate through a stratum of diseased tissue. I have a suspicion that if there had been an opportunity to study the case reported to-night by Dr. Morrison, it would have been found to present these peculiar psychic symptoms.

Dr. CHARLES K. MILLS.—A similar case of tumor of the brain I reported years ago to the Pathological Society of this city. In the size and position of the tumor, the case reported to-night reminds me of it. It was a fibroma, involving the pre-frontal lobe, and extending to the corpus callosum and the gyrus fornicatus, but not to the motor region. The patient showed this slowing of the mental reaction. After shouting at the man, we would have to wait sometimes a minute for his answer. As I put it at that time, it seemed to take a long time for an idea to get into his brain or to be appropriated by his brain. He also had motor symptoms on the opposite side of his body, which, however, were unlike the symptoms seen in tumor of the motor region. He had either a pressure or inhibitory paralysis which was not absolutely persistent. Hughlings Jackson holds the view that the prefrontal lobe is not only in a general sense concerned in higher mentation, but also in a certain sense it is a higher motor lobe. I believe that in such a case as that presented to-night, operation would almost certainly have resulted in death, unless great attention were paid to closing the dura mater and the scalp. One cause of death after such operations, as in porencephalus, is disturbance of the relation between external and internal pressure.

The PRESIDENT.—This tumor extended so very far back, that it is difficult to believe that there were no other symptoms than those recorded; yet when we look at the lateral surface of the brain, and see the marked difference in the course of the outer branch of the pre-central and also of the central fissures of either side, we see that the pressure has been exerted very gradually. This is probably the reason that there was no weakness on one side. This fact has of course been illustrated by a great variety of brain tumors. The Society may remember that, some years ago, I presented a large mass, almost as large as this, which started in the base of the second and first frontal convolution, and which pressed back the motor area to a great extent; but there had been no marked motor symptoms, the parts evidently having adapted themselves perfectly to a gradually increasing pressure. The mass extended back so far that certainly the caudate nucleus and the anterior limb of the capsule must have suffered, and if the growth had been rapid, other parts would have suffered from the transmitted pressure.

Dr. WHARTON SINKLER.—This tumor is very much like one that Dr. Catell will remember, the specimen of which I presented to the American Neurological Society. This was a sarcoma occupying a space just in the Rolandic fissure. It grew downward from the dura, and by pressure caused complete right hemiplegia. The patient also had some convulsions.

Dr. JAMES HENDRIE LLOYD read a paper entitled

ARTHROPATHY IN GENERAL PARESIS.[4]

He first briefly traversed the history of the subject of the arthropathy of tabes and other organic nervous affections, referring to the pioneer contributions of Dr. John K. Mitchell in 1831, Scott Allison in 1846, and of Charcot in 1868, and the more recent contributions of Ball, Clifford Albert, Rosenthal, and others. The different varieties of arthropathies were fully considered, and the arthropathy of Charcot discussed with reference to its occurrence in four diseases—namely, locomotor ataxia, syringomyelia, disseminated sclerosis, and general paresis. In a note, Dr. Lloyd referred to one case of possible insular sclerosis with arthropathy which was operated upon by Dr. J. W. Hearn for the removal of numerous foreign bodies of bone from the knee-joint. He believed that, in general paresis with arthropathy, either posterior sclerosis was present or there was good cause to suspect it. He regarded the arthropathies as trophic lesions and of spinal origin. The patient whose case was reported in detail, and which formed the basis of the contribution, belonged to Marie's hypertrophic type of arthropathy.

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[4] Dr. Lloyd's paper, which systematically discusses the subject of arthropathies, will appear in full in the second volume of the Philadelphia Hospital Reports.

J. D., aged thirty-eight, white; admitted to the wards in July. He had suffered for some years with pain in the legs, which had been called rheumatic. Otherwise he was apparently healthy up to one year and a half ago, when he had a swelling of his knees. At this time no mental symptoms were observed. Six months before admission he began to have mental symptoms; he had delusions of a persecutory tinge. He became silly and weak. Then he had a remission and was much better; then he relapsed, and was sent to the hospital. Drawling speech began three months before his admission. He had had no convulsions. There was no history of syphilis.

On admission he was very demented and dull, answering questions very imperfectly. He had a drawling speech and some unsteadiness of the facial muscles. There was very slight nystagmus; no intention tremor, no delusions could be demonstrated at first; the knee-jerk appeared to be abolished, but the knees were so swelled and deformed that it was difficult to test them. By tapping vigorously, a slight myotatic reflex was caused in the quadriceps. Both knee-joints were enormously enlarged; grating sounds could be heard in them easily. The bony structure on the anterior end of the condyles of the femur was enlarged, forming prominent bosses. Sensory tests were negative, because of the patient's state. The pupils acted on accommodation, but did not react to light—the Argyle-Robertson phenomena. Hearing was good; the right elbow was deformed by an old ununited fracture of the olecranon, but the man had fairly good use of the joint; the detached olecranon was easily movable and somewhat enlarged. The left forearm presented an extensive scar, the result of an old injury; the ulnar nerve had been cut across many years before, and there was the characteristic wasting and *main en griffe*.

A few days after admission the patient had a maniacal episode—so characteristic of paresis—which required that he be strapped in bed. He was very violent in his delirium, fighting and resisting, but showing no intelligence. After this episode he gave evidence of a grandiose delusional state. He was more demented, and talked in a rambling and incoherent way about finding an immense piece of silver and large sums of money. His speech became still more drawling and unsteady. Because of the deformity of his knees it could never be satisfactorily tested whether he swayed with his eyes shut. He managed to walk about. In the course of a few weeks incontinence of urine set in.

The joints grew more deformed. The patellæ were flattened and disfigured and increased in diameter. The bosses on the condyles were very prominent. No stalactites could be determined; grating sounds were always readily obtained; the joints were painless and free from heat or redness; there was evidently increase of fluid within them; no very marked œdema was seen around the joints, but still the skin had a very hard and tough feeling; there was increased lateral movement.

The electrical tests were of great interest. Very strong currents, both of faradism and galvanism, failed almost completely to cause contractions in the muscles of the thighs and legs. The only exception was in the peroneal group of the right leg, in which the reaction was normal. I think the reason for this failure was in the fact that the brawny and somewhat œdematous skin resisted the passage of the currents.

An examination of the eye grounds by Dr. Gould gave negative results.

Early in October the patient began to have retention of urine. This required the use of the catheter twice a day. Although every precaution was taken against sepsis, the urine soon became alkaline and contained pus. The bladder was washed out with a solution of boracic acid daily. On the 16th inst. the man had a severe chill, followed by a rapid rise in temperature. The chill was repeated daily. The patient's condition became grave. A diagnosis of infectious cystitis and pyelitis was made. He died on the 20th inst. with œdema of the lungs.

The post-mortem findings were as follows:

Brain.—The dura was very adherent to both the skull and the membranes. It was thickened. The longitudinal sinus was filled with a black clot. There was sub-arachnoid œdema. The frontal lobes especially were abnormal and in marked contrast to the rest of the brain. They were blanched, the arachnoid somewhat opaque, the convolutions evidently shrunken. The pia mater stripped easily from the brain in the Rolandic and parietal regions of the left hemisphere, but on the frontal lobe it adhered, especially over the exterior and extreme frontal region and along the gyrus marginatus. On the right hemisphere this condition was more marked and more extensive. Almost all these convolutions tore when the pia was stripped from them. The perivascular sheaths were filled with a white opaque fluid. At the base of the brain adhesions were very extensive, so much so that it was difficult to open up the Sylvian fissure, the opposing surfaces being agglutinated together. The pia-arachnoid was thickened and opaque and strongly adherent in other regions of the base. The large blood-vessels at the base were normal. On laying open the brain-axis the lateral ventricles were found to be dilated and full of a clear fluid in excess. The ependyma was not thickened, but normal in appearance. The aqueduct of Sylvius was pervious. The walls of the third ventricle were agglutinated together. The basal ganglia, internal capsules, mid-brain, pons, medulla, and cerebellum were normal to naked eye inspection.

The Knee-Joints.[5]—The right joint was as follows: The synovic membrane in front was of a steel-gray color, mottled with blue patches and covered with numerous small miliary calcareous nodules. The anterior edges of the condyles of the femur were greatly hypertrophied in nodular masses or rogosities. These formed bosses extending upwards and outwards about two centimetres beyond the edge of the condyle. In the anterior notch between the condyles was a triangular mass of nodules embedded in tissue and freely movable. These nodules were of stone hardness. Over the articular surface of the right condyle there was left the lining membrane of the joint considerably thickened and rough in some places, in others thin, and presenting about the centre an eroded patch. Over the surface of the left condyle, the lining membrane was absent, and the bone was worn and eroded, especially toward the periphery. There was a series of small eroded patches with nodules running along the outer border to the posterior extremity of the condyle. In brief, most of the soft structures of the joint showed destructive changes. The semi-lunar cartilages were much worn and eroded, and easily displaced. The inner surface of the capsular ligament showed some pedunculated masses. The crucial ligaments were apparently wasted, but still held firmly. The end of the tibia was covered with thin, worn and eroded membrane; part of the surface of the bone was bare. The joint contained a green, thick, opaque fluid. The patella was quite deformed. It was thin and flattened, with rugous edges. The under surface was covered with shreds of membrane. The capsular ligament was much distended and the joint cavity extended to an abnormal limit upon the front of the femur. The left knee-joint was rather larger and more distended than the right. The same general characteristics were to be noted—distended capsule, eroded membranes, nodular pedunculated formations, and bony bosses on the anterior edges of the condyles.

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[5] Very accurate plaster casts were made of the joints by Dr. H. G. Cattell, Assistant Pathologist of the Hospital, and are now in the Museum.

Kidneys and Bladder.—The kidneys were large; the capsules adherent. The pelvis of the right kidney was enormously dilated and filled with a purulent fluid. The ureters were dilated. The bladder was thickened and presented evidence of a recent catarrhal process.[6]

DISCUSSION.

Dr. CHARLES K. MILLS.—This patient was in my wards also, and I had an opportunity of studying him a few weeks before death, and also just before death. It was certainly a most interesting case of arthropathy, and particularly on account of the paretic dementia which was present. As to this question of paretic dementia and the occurrence of arthropathies in the course of the cerebral affection,. I think that one of the expressions which Dr. Lloyd used might be misleading. He spoke of these cases and other cases as belonging to the ascending type of posterior sclerosis. One of the earlier views of paretic dementia, when it was the termination clinically of posterior sclerosis, was that the degenerative conditions in the spinal cord continued through the motor tracts all the way to the cerebrum and to the cerebral cortex. This is certainly not the correct view. A number of years ago I had for several years a case of posterior sclerosis under my care in private practice. The patient became paretic and went to the Pennsylvania Hospital for the Insane, and subsequently to Danville, where he died. The body was sent to Philadelphia, and a postmortem was made, and twenty or thirty sections from the cord, and all the way to the cortex, were examined under the microscope. Similar cases have been recorded. The disease perhaps ascends so far as the cord is concerned; but the cerebral condition is only a localized expression of a general condition. The disease does not usually extend anteriorly beyond the oblongata and pons. This has a little bearing upon the discussion of this case. We are not to regard these arthropathies as having anything to do with the paretic dementia. They are connected with the spinal lesion. The muscles were not extremely atrophied, and there is not much doubt that the absence of electrical reaction was due to the condition of the skin and subcutaneous tissues. Comparing the skin of the arm and hand with that of the thigh and leg, the difference was very marked; a solid mass, quarter to half an inch thick, seemed to be present under the skin of the thighs.

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[6] Acknowledgments are due to Dr. H. D. Beyea and Dr. Joseph Sailer, resident physicians, for valuable clinical work on this case. The specimens will be examined microscopically.

Dr. Lloyd refers to the occurrence of arthropathies in cases of hemiplegia, and in spinal affections other than posterior sclerosis. The arthropathies in cerebral hemiplegia are not to be explained in the same way as those of these cases. One explanation of them is that they are due to neuritis. They are due neither to the cerebral lesions, nor, strictly speaking, to the secondary changes in the motor tract, but to the peculiar conditions presented in hemiplegia, the relaxation of the joints and the traumatisms to which the limbs are subjected, and to the fact that the relaxation is itself in a certain sense a traumatism. As a result of the traumatisms inflicted by the flaccid, dangling, or sometimes contractured arm, neuritis is excited, involving a large part of the arm only, and sometimes the joints as well.

Dr. Lloyd referred to disseminated sclerosis with arthropathies. Of this I have seen one or two instances. In amyotrophic lateral sclerosis, I have seen peculiar painful and semi-anchylosed conditions of the joints, one within a few weeks, in the case of a lady who for three or four years has been developing symptoms of progressive muscular atrophy, or, more properly, of amyotrophic lateral sclerosis. Both shoulder-joints, but one more than the other, have been subject to attacks of pain without much swelling, but now there is a certain amount of adhesion.

Dr. WHARTON SINKLER.—I agree with Dr. Mills that the arthropathies of hemiplegia are quite different from those of spinal origin. Dr. Lloyd refers to the fact that in some of the benign cases of arthropathies in tabes, recovery may take place in the joint. There is an interesting example of that in the wards of the Philadelphia Hospital. The patient has suffered from both epilepsy and locomotor ataxia, and at one time he had enormously enlarged knee-joints and was so ataxic that he could scarcely stand. I looked him up a short time ago, intending to show him in the clinic with some other cases of spinal arthropathies, but found that the knees had recovered and that he had regained the power of coordination to such an extent, that instead of walking with crutches he used only a cane.

I would like to refer to another patient at the Philadelphia Hospital who has remarkable spinal arthropathies and who is suffering from none of the forms of spinal disease referred to to-night. The patient has chronic myelitis, with contractures, exaggerated knee-jerk and trophic lesions. He had an enormous arthropathy of his shoulder, the joint at one time filled with fluid. Crepitation was also present. It was never painful. During the past year this swelling has very much subsided.

The PRESIDENT.—Some years ago I had a photograph made of the first patient to whom Dr. Sinkler has alluded. In addition to the enlargement, the knees were exceedingly retroflexed. There was no lateral displacement. Last winter when I wished to demonstrate the condition to my class, I found that while there was still some retroflexion, it had markedly diminished.

In the second case mentioned by Dr. Sinkler, the patient has a delusion that all his troubles are due to poisoning with arsenic. In this case there is loss of the temperature-sense over the entire right arm and side of the face, and it is probable that we have not a case of myelitis, but of syringomyelia with arthropathies in the shoulder-joints. In syringomyelia the arthropathies are more common in the upper extremity, while in locomotor ataxia they are more frequently met with in the lower extremity.

Dr. JAMES HENDRIE LLOYD.—I think that Dr. Mills may have misunderstood one point in my paper. I did not say that the joints in hemiplegia are like spinal arthropathies. I only referred to an historical fact. They were originally described by an Englishman, Scott Allison, in 1846, at which time the question came up as to their possible rheumatic origin. They are pseudo-rheumatic at least. They are painful and present some of the symptoms seen in rheumatic joints. They are entirely distinct from spinal arthropathies. So the arthropathies of acute myelitis are apt to be of this same pseudo-rheumatic character, as in Gull's case and as in Dr. John K. Mitchell's case, which suggested to the latter his theory of the central origin of rheumatism. In 1831 he published a paper on this subject in the "American Journal of the Medical Sciences." These are different from the non-painless spinal arthropathies.

I have no doubt that in the case reported this evening, the connection of the joint disorder is with the cord, but it will require the microscopical examination to decide that. I do not care whether you call this case an example of the ascending type of general paresis or not. I have never believed that the degeneration in so-called ascending cases of general paresis begins in the cauda equina and extends upward. There is, however, sometimes a preponderance of posterior sclerosis in the earlier stages, which posterior sclerosis precedes the onset of the general paralysis. I have seen cases where the symptoms of locomotor ataxia existed for some years before the onset of the paretic dementia.

The PRESIDENT.—I might recall the fact that some years ago we had a collection of bones and joints exhibited to the Society. At that time I showed some bone sections. I found in some of the smaller joints of the foot that the cavities were obliterated and that bone tissue had been thrown across the joints. It is interesting, therefore, to note that the irregular deposit of bony tissue is not merely (at least in some instances) a calcareous deposit, but is made up of true bony tissue.

Dr. CHARLES W. BURR read a paper entitled

TUMOR OF RIGHT PREFRONTAL LOBE.

R. G., male, married, American, aged forty-four, box-maker, came to Dr. Sinkler's clinic at the Infirmary for Nervous Diseases, December 3, 1888, presenting the following history: During the summer he had suffered much from vertigo, thought to be due to dyspepsia. About August 1st, while standing in a street-car, his legs suddenly gave way beneath him and he fell. He was assisted home and put to bed, after which he became comatose and remained so the entire night. There was no convulsion. The next morning he was perfectly conscious, got up, dressed, complained of severe headache, and vomited several times. The third day he returned to his work and continued at it for three weeks. Six weeks later he had another severe attack, which was thought, by the physician who attended him, to be uræmic. On recovering consciousness, he saw double. He has not worked since. Diffuse headache and vertigo have continued. He has had one more attack of coma. Vision has been failing for several months. He uses alcohol very moderately. Syphilis is denied, and evidence of it cannot be found.

Present State.—Well built, healthy-looking man; gait good; station good; no ataxia in legs or arms; no paralysis; knee-jerks plus (later they were diminished); no impairment of taste or smell; hearing apparently normal, though no careful test was made; no pain on percussion of scalp; general sensation normal; mental condition good; no loss of memory; no slowing of mental activity; speech normal. Dr. Geo. E. De Schwinitz reported the eye condition as follows: "Discs swollen; R. E. + 6, numerous, tortuous, transverse veins pass over outer side. L. E. + 7 D. at apex; edges + 6 D. Irregularly contracted fields; lateral nystagmus." Urine free from albumen and sugar.

Further Course of Disease.—By February, 1889, there was complete blindness. From this time on the attacks of unconsciousness slowly increased in frequency, until finally two or more occurred every week. While his wife alleged that convulsive movements occurred in some of these attacks, this was never the case when I was present. The onset was always sudden; the coma complete; the respiration stertorious; the face congested;

there was persistent and obstinate constipation. During the course of the disease the patient put on much fat. In the last six or eight weeks of life his mind became sluggish and he wandered slightly in speech. He died September 14, 1891, three years after the onset of the symptoms.

Remarks.—The headache, vertigo, attacks of coma, choke disc, and nystagmus rendered the diagnosis of intra-cranial tumor very easy. Its localization, however, was a matter of extreme difficulty, and the decision I finally made that the cerebellum was probably involved proved post-mortem to be false. I was largely influenced by the fact that in the absence of paritive localizing symptoms, nystagmus, with choke disc and complete blindness, point to cerebellar disease. I explained the absence of cerebellar ataxia by non-involvement of the middle lobe. I think the error was excusable, or rather, the graver error was in attempting localization without sufficient data.

Autopsy was made nineteen hours after death, the body having been kept on ice. Rigor mortis was marked. The scalp was not adherent to the calvarium. The latter was very thin, and upon its inner surface were many worm-eaten depressions filled by small cauliflower-like excresences from the dura. The dura was congested, and over the right frontal region firmly attached to the bone and thickened. On removing the dura a softened mass attached to it came away from the outer aspect of the right prefrontal lobe. A harder mass was left behind, involving the entire lobe except the median cortex and the orbital surface. It did not extend into the ascending frontal convolution. Microscopically it presented the appearance of a round-celled sarcoma. In the softened part hemorrhage was present. The optic nerves were much shrunken.

SECONDARY CANCER OF THE DURA INVADING THE CEREBELLUM.

M. G., female, white, single, aged about forty-five years. In March, 1891, the left breast was amputated for carcinoma; and in April of this year the left axilla was opened, to remove cancerous glands. Immediately after this operation the left arm was found to be palsied. Soon after she began to complain of rapidly increasing weakness, and had an attack of unconsciousness without convulsion. There was at times slight frontal headache; never vomiting. I saw her once only, a couple of weeks before death, while she was under the care of Dr. Harvey Shoemaker, to whom I am indebted for permission to show the specimen. At that time she was emaciated, cachetic, and bed-ridden. There was no palsy of legs or arms except that due to the local injury; the lower face was palsied; tongue protruded straight; there was distinct inco-ordination of movement; the knee-jerks were much diminished; sensation was normal; there was constant lateral nystagmus: the pupils were equal and reacted to light. Acuteness of vision could not be measured; she could, however, see apparently quite well. Dr. Cross reported that there was no fundus lesion; urine was normal. She was apathetic, and almost never spoke except when addressed; she would answer all questions asked her, but soon became mentally fatigued, and then would reply at random. For some weeks before death she had attacks of sudden oncoming unconsciousness without convulsion; duration varied from one-half hour to several hours.

She died in June, 1892. Diagnosis: Intra-cranial cancer involving the cerebellum.

Autopsy.—A hard mass, about as large as an English walnut, was found growing from the dura mater at the posterior edge of the right lobe of the cerebellum. It had invaded the cerebellar tissue, and was surrounded by an area of softening involving the whole interior of the lobe. Microscopical examination showed it to be carcinomatous.

DISCUSSION.

Dr. WHARTON SINKLER.—This patient was a regular attendant of my clinic for nearly three years. At first, the diagnosis of brain tumor was readily made, but the difficulty in localizing the growth was exceedingly great. There were absolutely no focal symptoms. There was a history of a convulsion, but there was some doubt whether or not there had been any convulsive movements. In the subsequent attacks there was simply stupor. There was no pain, no headache, no loss of memory, no impairment of the special senses. No pain could be elicited by percussion. Therefore, from the standpoint of the neurologist as well as from the standpoint of the ophthalmologist, it seemed probable that the growth was located posteriorly.

The President.—It is exceedingly interesting to note that there were special attacks of stupor, which attacks seemed to replace the convulsions which are seen when the tumor involves the motor area. They seemed to be periodic attacks of irritation, causing, as their focal symptom, stupor. This certainly suggests a point for future use in diagnosis. If, as was claimed, the man really had a general convulsion, it was probably due to irritation of the dura, and not focal in character. The fact that the base of the second frontal gyri is pressed upon would be in harmony with the history of nystagmus. The case is also of interest as illustrating the difficulty of distinguishing between cerebral and cerebellar tumors. In a case at the Philadelphia Hospital, studied by Dr. Mills and myself, it was impossible to make a positive diagnosis. There was general headache, and, as it was increased by percussion over the frontal region, we thought that the tumor might be in that position. Exploratory operation failed to reveal the growth, and the post-mortem demonstrated that it was in the cerebellum.

Dr. Wharton Sinkler.—In the case referred to by Dr. Lloyd, the supposition of general paresis was justifiable from the circumstances. The man came into the house with distinct delusions of grandeur. He could walk a thousand miles; he had enormous strength; he had a large amount of money in his box at some sailors' boarding-house. These were the prominent symptoms on admission. Later, the unsteadiness of gait, pitching backward, and eye lesions were observed.

Dr. Charles K. Mills.—One affection that should be borne in mind in considering the question of disease of the cerebellum, and that is labrynthine disease. The two affections sometimes resemble each other pretty closely. Of course, in labrynthine disease there is usually tinnitus and deafness, but you may have both in disease of the cerebellum. In labrynthine disease another symptom may be present, which is occasionally seen in cerebellar disease—that is, lost knee-jerk. I think that this may be explained in a method similar to that by which Jackson explains the absence of the knee-jerk in cerebellar disease itself.

Adjourned.